Homing of Adult Chinook Salmon after Brief Exposure to Whole and Dispersed Crude Oil

School of Fisheries WH-10, University of Washington
Seattle, Washington 98195, USA

CLAYTON D. MCAULIFFE
Chevron Oil Fields Research Company, La Habra, California 90631, USA

Abstract. -- Adult chinook salmon Oncorhynchus tshawytscha that had returned to the University of Washington, Seattle, hatchery were exposed for 1 h to either whole Prudhoe Bay crude oil, a chemical dispersant, or chemically dispersed oil in fresh water. The oil exposure concentrations were higher than under oil spill conditions measured in the field. Members of the treatment groups and similarly handled controls were held for 1 d after exposure and then displaced downstream. Neither frequency of homing (72% overall) nor days to return to the hatchery (mean = 3.2 d) were affected by the treatments. Retention of some treated fish at the hatchery determined that longevity was sufficient to prevent significant bias in estimates of homing. Later in the season, homing speed increased and longevity decreased, but homing frequency remained relatively constant.

Adult Pacific salmon Oncorhynchus spp. are known to return to their natal streams to spawn, guided in the final stages of their homing migration by olfactory cues (reviewed by Brannon 1982; Hasler and Scholz 1983). As offshore oil exploration has increased, so has concern that an oil spill might interfere with salmon homing. Experiments have indicated that juvenile salmon avoid the water-soluble fraction of crude oil (Rice 1973) or a model mixture of monocyclic hydrocarbons (Maynard and Weber 1981). However, under certain circumstances, juvenile salmon do not avoid whole crude oil (Morrow 1973; Maynard 1980).

Weber et al. (1981) reported that adult salmon migrating upstream avoided a model mixture of monocyclic hydrocarbons at concentrations of 3.2 mg/L and higher. While avoidance of a major oil spill might delay or detour migrating salmon, petroleum hydrocarbons might also reduce olfactory sensitivity, as some other pollutants seem to do (Hara et al. 1976), thereby preventing the discrimination of the natal stream crucial to homing.

Because a variety of studies have indicated that chemically dispersed oil may be more injurious than whole crude oil (Linden 1975, 1976; Wells and Keizer 1975; McKeown and March 1978), we hypothesized that dispersed oil might hamper homing more than whole oil. In addition, some dispersants are known to damage fish chemoreceptors (Bardach et al. 1965; Cancalon 1983) or diminish chemosensory responses (Hara and Thompson 1978; Olsén and Höglund 1985). We designed the present study to determine whether brief exposure to whole or dispersed crude oil or dispersant alone affects the homing ability of adult chinook salmon Oncorhynchus tshawytscha.

Methods

The chinook salmon used in the experiments were part of the University of Washington population that returns to the Seattle hatchery in October and November (Whitman et al. 1982). The mature males used (average fork length, 62 cm; average weight, 2.7 kg) were in good condition because they had ascended only 9 km of slow-moving fresh water to reach the hatchery. Between October 9 and November 6, 1981, 272 chinook salmon were seined from the hatchery pond, anesthetized with tricaine (MS-222), and tagged with numbered, color-coded Petersen discs. After a 1-h recovery period, five or six fish were placed in each of four open 1.5-m circular tanks holding 1,300 L of aerated, circulated lake water. One tank consisted of the untreated control, the second received 913 mL of Prudhoe Bay crude oil, the third received 13.7 mL of a chemical dispersant mixture (44.5% Tween 85, 11.0% Span 80, and 44.5% solvent), and the fourth received 137 mL of crude oil and 13.7 mL of the dispersant mixture. These concentrations were selected to be somewhat

1 Contribution 703 from the School of Fisheries, University of Washington.
higher than concentrations expected under field conditions (discussed later).

Uniformly mixed, the 913 mL of untreated oil would have produced 702 µL oil/L in the tank. Had the slick been uniform, it would have formed a 0.5-mm-thick layer. If the dispersant-treated oil had been uniformly mixed, it would have produced a concentration of 105 µL oil/L. Total oil was determined for all exposure tanks by carbon tetrachloride extraction and infrared (IR) examination of 1-L water samples drawn from mid-depth at the end of the exposure periods. Similarly, C₃-C₁₀ hydrocarbons were determined by a gas equilibration method (McAuliffe 1971, 1979, 1980) on water samples collected during the 1-h exposure.

Heavy plastic screens were suspended 5 cm below the water surface in all tanks to prevent the fish from jumping out or directly contacting surface oil. After 1 h, the surface oil was removed with absorbent pads and the fish were returned to a holding raceway containing clean hatchery (lake) water.

Homing experiment.—One day (20–22 h) after exposure, fish from all four treatment groups were simultaneously transported about 5 km downstream and released. In total, 215 fish were released on 13 separate days. The hatchery pond was seined for returns three times weekly and visual checks of color-coded tags on fish in the pond on the other days determined return times to the nearest day. The number of chinook salmon homing and the average time to return for each of the experimental treatments were compared to evaluate the effects of exposure.

Longevity experiment.—Unequal return percentages among the four groups could be caused by differential mortality as well as by effects on olfaction and homing. Therefore, to establish whether time to mortality influenced apparent homing success, 14–15 chinook salmon from each treatment group were held in a 9.1 x 1.8-m raceway containing clean hatchery water between October 9 and November 16. Approximately 20 fish were held in the raceway at any time, and as individuals died they were replaced to maintain the same population density. We used 57 chinook salmon in this experiment.

Results

Homing Experiment

Of 215 chinook salmon displaced downstream, 154 (72%) returned to the hatchery pond (Table 1). The proportions of returns from the four experimental groups were not statistically different ($X^2$ for heterogeneity = 1.32; 3 df; $P > 0.05$). The average return time was 3.2 d with the treatments having no detectable effect on homing speed ($P > 0.10$; Kruskal-Wallis test). Although fish released later in the season tended to return faster than those released earlier, the frequency of return remained relatively constant (Figure 1).

Eight of the 61 chinook salmon that did not return to the hatchery were recovered elsewhere: two were caught by sport fishermen, one was caught in a commercial gill net, and one was found dead. Four chinook salmon entered a National Marine Fisheries Service hatchery across the ship canal from the University hatchery. However, there was no indication that straying was influenced by treatment, as two of them were controls, one had been exposed to dispersant, and one to dispersed oil.

Longevity Experiment

The longevity experiment determined whether or not mortality influenced apparent homing suc-

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**Table 1.**—Frequency of homing and time to return to the hatchery by chinook salmon subjected to various treatments of oil and a dispersant.

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Number released</th>
<th>Number returned</th>
<th>Days to return (mean±SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>53</td>
<td>35 (66%)</td>
<td>4.0±2.9</td>
</tr>
<tr>
<td>Oil</td>
<td>54</td>
<td>41 (76%)</td>
<td>2.7±2.1</td>
</tr>
<tr>
<td>Dispersant</td>
<td>54</td>
<td>39 (72%)</td>
<td>3.5±3.0</td>
</tr>
<tr>
<td>Oil + dispersant</td>
<td>54</td>
<td>39 (72%)</td>
<td>2.8±2.1</td>
</tr>
<tr>
<td>Total</td>
<td>215</td>
<td>154 (72%)</td>
<td>3.2±2.6</td>
</tr>
</tbody>
</table>

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**Figure 1.**—Average time from displacement to return (homing) and average percent return for chinook salmon in all treatment groups. Dates are those of downstream release.
cess. Fish that arrive early in the season tend to live longer than those from the end of the run, and this was evident in this experiment (Table 2). On average, the 57 chinook salmon lived 10.6 d in the holding raceway (Table 3). These results do not include one fish from the dispersed-oil treatment that died during exposure and one each from the oil and dispersed-oil treatments that died within 1 d of exposure. These were excluded because chinook salmon that died within 1 d of exposure would not have been released for homing.

The average numbers of days to mortality were adjusted because chinook salmon from the four treatments were not maintained in the holding raceway in constant proportions and because date of treatment was correlated with longevity (Table 3). Bonferroni multiple comparisons were made on all six pairs of adjusted longevity means for the four treatments (Weisberg 1985). The only significant ($P < 0.05$) difference was between the control and oil + dispersant treatments.

Analysis of the homing and mortality schedules demonstrated that mortality had a relatively minor influence on return success. By the sixth day after treatment (fifth day after release), 87% of the fish that eventually homed had done so, but only 9% of the fish in the raceway had died (Figure 2).

**Hydrocarbon Concentrations**

At the end of the 1-h exposure periods, the mean concentrations of total hydrocarbons measured by IR were $0.18 \pm 0.11 \mu L/L$ in the oil treatment and $21 \pm 7 \mu L/L$ in the dispersed-oil treatment. The C$_1$–C$_{10}$ hydrocarbon analyses revealed that undispersed oil on the surface slowly introduced dissolved hydrocarbons to the water and concentrations were still increasing at the end of 1 h (Figure 3). In contrast, total C$_1$–C$_{10}$ concentrations in the dispersed-oil tanks reached a peak after 10–15 min and then decreased.

**Discussion**

The relatively high return frequency in all groups indicates that 1-h exposure to whole or dispersed crude oil or to dispersant did not hamper chinook salmon homing. Return to the hatchery pond represents a meaningful choice by the fish, as the hatchery contributes only about 0.07 m$^3$/s to the ship canal's total flow of 20–25 m$^3$/s. Hasler and Scholz (1983) reviewed the evidence that olfaction is essential to homing. Furthermore, unpublished experiments at the University of Washington have shown that displaced chinook salmon with their nares plugged do not return to the hatchery. It appears that anosmic fish do not follow conspecifics back to the hatchery. We therefore conclude that the olfactory systems were not impaired. Thus, although adult chinook salmon might avoid oil (Weber et al. 1981), brief exposure to whole or dispersed oil did not prevent or delay homing. For comparison, Weber et al. (1981) reported that migrating salmon avoided hydrocarbon concentrations of 3.2 mg/L whereas the highest concentration that our fish were exposed to was about 1.8

<table>
<thead>
<tr>
<th>Exposure dates</th>
<th>Sample size</th>
<th>Days to mortality (mean±SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Oct 9–14</td>
<td>15</td>
<td>13.7±2.6</td>
</tr>
<tr>
<td>Oct 16–21</td>
<td>8</td>
<td>11.5±2.4</td>
</tr>
<tr>
<td>Oct 23–28</td>
<td>14</td>
<td>10.3±3.8</td>
</tr>
<tr>
<td>Oct 30–Nov 4</td>
<td>11</td>
<td>8.1±5.0</td>
</tr>
<tr>
<td>Nov 6</td>
<td>9</td>
<td>8.4±1.1</td>
</tr>
</tbody>
</table>

**Table 3.**—Average longevities of chinook salmon exposed to various treatments of oil and a dispersant.

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Sample size</th>
<th>Unadjusted longevity (±SD)</th>
<th>Adjusted longevity for arrival date</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>15</td>
<td>13.1±2.4</td>
<td>12.7</td>
</tr>
<tr>
<td>Oil</td>
<td>14</td>
<td>10.7±3.5</td>
<td>10.4</td>
</tr>
<tr>
<td>Dispersant</td>
<td>14</td>
<td>10.1±3.0</td>
<td>10.2</td>
</tr>
<tr>
<td>Oil + dispersant</td>
<td>14</td>
<td>8.6±5.1</td>
<td>9.0</td>
</tr>
<tr>
<td>Total</td>
<td>57</td>
<td>10.6±3.9</td>
<td></td>
</tr>
</tbody>
</table>

**Figure 2.**—Cumulative proportion of the total number of homing chinook salmon that returned in the days following treatment, and cumulative proportion of chinook salmon held at the hatchery that died on those days (all treatment groups combined).
mg/L (Figure 3). The dispersant concentration of 10.5 μL/L also produced no apparent effect on subsequent homing behavior. For comparison, Olsson and Höglund (1985) reported that the chemosensory responses of juvenile Arctic char Salvelinus alpinus were somewhat diminished after a 96-h exposure to 1-2 μL detergent/L.

In a recent review, Brown et al. (1982) discussed several classes of effects of pollution on fish chemoreception, but, in general, the effects were associated with exposure for days or weeks. The brief exposure to oil in the present study was selected because adult chinook salmon would probably not remain under an oil slick for a long period. Sockeye salmon Oncorhynchus nerka (typically smaller than chinook salmon) swim about 2 km/h in coastal waters (Madison et al. 1972; Quinn and terHart, in press). Therefore, they would not be exposed to oil for more than a few hours under any but the largest spills.

It should be noted that, in our experiments, direct exposure of the nares to surface oil was prevented by the screen under the water's surface. When migrating through coastal waters, sockeye salmon may spend large amounts of time near the surface (e.g., Quinn and terHart, in press) and can often be observed jumping in nearshore areas. It remains to be determined whether an oil spill would affect the depth distribution of salmon in open water and whether they would jump through surface oil.

The treatment concentrations of Prudhoe Bay crude oil, dispersed oil, and dispersant were selected from published data on the possible concentrations salmon might encounter in a near-shore oil spill. Theoretical calculations of oil slick spreading and areal measurements of known oil spill volumes (McAuliffe et al. 1981) show that average thickness of oil slicks is about 0.1 mm, about one fifth of the thickness of the oil in the exposure tank.

In a series of chemically dispersed oil slicks (Cormack and Nichols 1977; McAuliffe et al. 1981), the highest total oil concentration measured by IR from a dispersed Prudhoe Bay slick was 41 μL/L at 1 m depth 15 min after aerial spraying. This decreased to 12–14 μL/L from the surface through 6 m depth (2.3 μL/L at 9 m) 54 min after spraying (see also review by Chapman 1985). After 3.5 h, the highest concentration was 2 μL/L. The dispersed-oil exposure for chinook salmon was 21 μL/L, higher than would be expected under field conditions. The C₁₀–C₂₀ hydrocarbons averaged about 0.075 mg/L during the first hour after aerial spraying in a study of a Prudhoe Bay crude oil spill off southern California (McAuliffe et al. 1981). Thus the chinook salmon, exposed to about 1.5 mg/L (Figure 3), experienced roughly 20 times the concentration seen in the field. The C₁₀–C₂₀ hydrocarbon concentrations under untreated oil slicks did not exceed 0.002 mg/L.

In the present study, the experimental oil exposure occurred in fresh water, not seawater as would be the case with a nearshore marine spill. The sensitivities of young salmon to oil are different in fresh and salt waters (Rice 1973; Rice et al. 1975). Because oil appears to affect ion transport in the gills (Morrow et al. 1975; McKeown and March 1978), the salinity of the water may influence the effects of oil. However, a study paralleling the methods of the present study (albeit with a smaller number of returns) found no evidence that whole or dispersed oil affected homing by coho salmon Oncorhynchus kisutch in seawater (Nakatani et al. 1985).

Acknowledgments

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References


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